

ABSTRACTS

Abstracts for Chaired Posters:

CP5.

INVOLVEMENT OF AUTOPHAGY IN THE EFFECT OF EXERCISE ON LEFT VENTRICULAR HYPERTROPHY INDUCED BY HIGH FAT DIET IN RATS

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Objectives: Left ventricular hypertrophy (LVH) associated with obesity increases the morbidity and mortality of cardiovascular disease, which could be attenuated by exercise in overweight and hypertensive patients. The lysosomal degradation pathway – autophagy is reportedly mediated the beneficial effect of exercise on glucose and lipid homeostasis. The present study aimed to investigate the involvement of autophagy in the effect of exercise on LVH induced by high fat diet in rats.

Methods: Female adult SD rats were divided into 4 groups namely: (i) high fat diet (HFD), (ii) HFD+exercise, (iii) exercise, (iv) control. Rats in the HFD groups were orally fed with high-fat chow (30% fat) daily for 12 weeks, and rats in the exercise groups had exercise with a motorized wheel in the last 4 weeks. Noninvasive measures of systolic pressure and fat composition were assessed, respectively by tail cuff and MRI. The expression of markers for cardiac hypertrophy and the protein expression in autophagic pathway were determined by quantitative real time-PCR and western blot, respectively. Statistical significance was at $p < 0.05$ with ANOVA analysis followed by post-hoc tests.

Results: Rats fed with HFD had LVH (increased heart weight and LV/RV+septum ratio) with higher levels of body weight, arterial pressures and fat composition than that of the control rat. In addition, the QTc interval and

the diameter and disarray of ventricular myocytes were significantly increased in the HFD group, supported by elevated levels of the expression of hypertrophic markers (ANP, BNP, β -MHC). These parameters were attenuated by exercise in the HFD-fed rats. Moreover, we found elevated levels of LC3II in the HFD heart, which were also attenuated by exercise, suggesting an involvement of autophagy in the beneficial effect of exercise. Furthermore, the expression level of AMPK α was also increased in the exercise groups.

Conclusion: We demonstrated that exercise lowers the body weight and attenuates the HFD-induced LVH in rats, which probably involves autophagy. Future studies will focus on the role of autophagy in the pathogenesis.

CP6.

THE FUNCTIONAL ROLE OF TRPV4 CHANNELS IN BARORECEPTOR SENSITIVITY

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There are two major arterial baroreceptors that can detect blood pressure change in the arteries. They are the aortic (arch) baroreceptor and carotid (sinus) baroreceptors. Aortic baroreceptor detects blood pressure in the aorta. Carotid baroreceptor detects blood pressure in carotid artery, which carries the blood to the brain. Reports suggest that there are differences between these two baroreceptors in terms of their pressure sensitivity and functional role. But the results are controversial. In the present study, the properties of the two baroreceptors were investigated. It was found that aortic baroreceptor neurons were more sensitive to pressure change than carotid baroreceptor neurons. Electrophysiology studies showed that, compared to the carotid baroreceptor neurons, a higher percentage of aortic baroreceptor neurons were stretch-sensitive. Furthermore, the pressure threshold that can initiate action potential firing was found to be lower in the aortic baroreceptor neurons than in the carotid baroreceptor neurons. Uniaxial stretch-induced $[Ca^{2+}]_i$ rise was compared between aortic and carotid baroreceptor neurons. Again, the aortic baroreceptor neurons were found to be more sensitive to stretch than the carotid baroreceptor neurons. Immunostaining experiments revealed that, compared to carotid baroreceptor neurons, a much higher percentage of aortic baroreceptor neurons were TRPV4-positive.

Consistently, the stretch-induced $[Ca^{2+}]_i$ rise could be inhibited by RN1734, which is a potent TRPV4 channels blocker. Taken together, these results suggest that the difference in pressure sensitivity between the aortic and carotid baroreceptor neurons was probably brought about by TRPV4 channels, which are stretch-activated channels.